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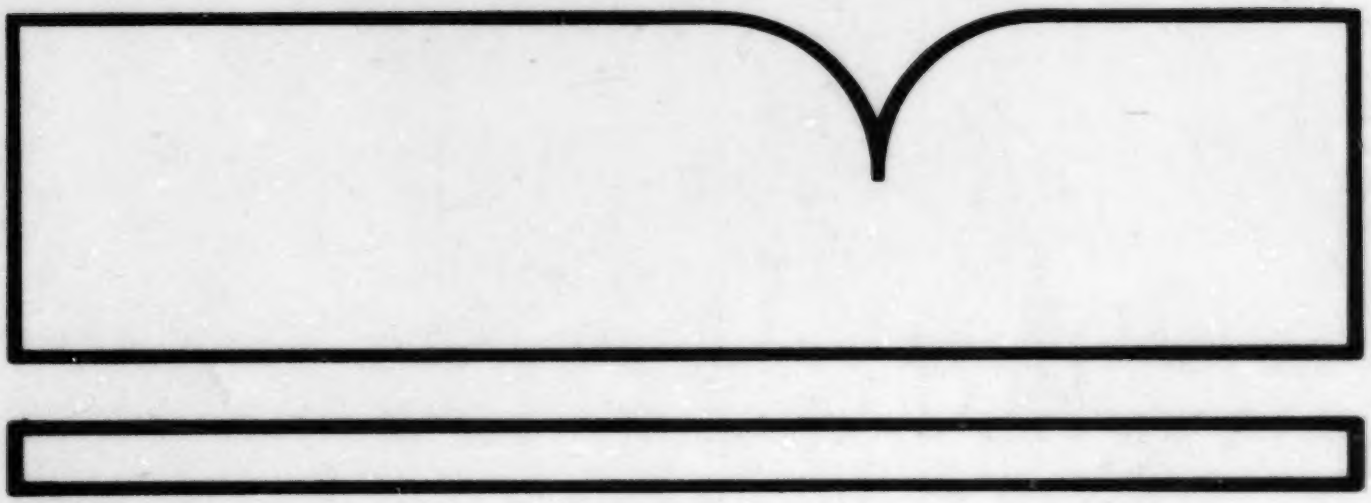

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16. ABSTRACT

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Passive Smoking and Height Growth of Preadolescent Children

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The attained height and height growth rate of 9273 children participating in a longitudinal study of the health effects of air pollutants were analysed to assess the association between passive exposure to cigarette smoke and physical growth between 6 and 11 years of age. Children were measured annually for 2 to 6 years. Each height measurement was adjusted for sex and age by the NCHS anthropometric standards. Each child's adjusted heights were then re-expressed as level of attained height and growth rate. Attained height exhibited a dose-response relationship with amount of current maternal cigarette smoking ($p<0.001$). Children whose mothers smoked ten or more cigarettes daily were approximately 0.65 cm shorter than childen of non-smokers, while children whose mothers smoked between 1 and 9 cigarettes per day were 0.45 cm shorter. However, passive smoking was not correlated with the child's growth rate. Exposure to paternal smoking was not significantly associated with height, either in terms of attained level or growth rate. These results indicate that passive smoking in the 6- to 11-year-old child does not continue to affect the growth rate of height and that the observed association between attained height and maternal smoking behaviour is due to exposures *in utero* and/or during infancy and the preschool years.

Several studies have demonstrated that passive smoking during childhood is associated with an increased risk of respiratory symptoms and reduced pulmonary function level.[1,2] However, few have asked whether the height of children also may be affected by passive smoking. There is substantial evidence[3] that smoking during pregnancy results in newborns that are both lighter and shorter (1.4 cm difference between newborns of smokers and non-smokers), even after taking gestational age differences into account.[4] Studies by Wainwright[5] of mothers who smoked during only one of two pregnancies discount earlier theories[6] that some inherent difference in certain women causes them both to smoke and to produce smaller babies. Studies by Miller *et al*[7] and Dunn *et al*[8] demonstrate that small (both short and light) infants born to smoking mothers do not show early catch-up growth. Butler and Goldstein[9] have suggested that these birth differences persist for many years and are dose-related. Eleven-year-old children of mothers who smoked more than 10 cigarettes per day during their fourth month of pregnancy were estimated to be 1.5 cm shorter than children born to non-smokers, while mothers who consumed 1 to 9 cigarettes per day had children who were 0.9 cm shorter. Persistent deficits between offspring of smokers and non-smokers noted by other authors include 0.9 cm at 5 years by Wingerd and Schoen,[10] 1 cm at 7 years by Hardy and Mellits[11] and 0.6 cm at 7 years by Davie *et al*.[12] One possible explanation for these differences is that *continued* exposure to sidestream smoke beyond birth is partially responsible for the persistence of the observed differences at birth. The availability of repeated height measurements on 9273 school children participating in a study of the health effects of air pollution along with information on their parent's smoking habits permits further study of these issues.

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DATA AND METHODS

Data

As part of an ongoing study of the health effects of air pollution, the Harvard Six City Study requires annual examination of participating school children and completion of questionnaires by their parents. The design of the study and the selection of the communities have been previously described by Ferris *et al*.[13]

Between 1974 and 1981, 13853 children were

examined in six cities across the United States—Watertown, MA; Kingston and Harriman, TN; Steubenville and Mingo Junction, OH; a geographically defined section of St Louis, MO; Portage, WI and several surrounding communities; and Topeka, KA. These cities were selected to represent a range of air quality based upon their historic levels of outdoor pollution. In each city, the initial examinations included all first and second grade school children in both the public and private schools, except Topeka in which a random sample of 50% of the schools was selected. Each year following the initial examination, the same children were seen again and a new first grade was added until approximately 1500 children were enrolled in each community.

For this analysis, 9273 white children who had at least two annual height measurements between the ages of 6 and 11 years were selected. A total of 3481 children were seen at age 6, 6682 at age 7, 7910 at 8, 7929 at 9, 6741 at 10, and 4817 at age 11. The median number of visits per child was four (with a mean of 4.04). Standing height was measured in stockinged feet. Other data collected included annual reports of the numbers of cigarettes smoked daily by mother and father, self reporting of smoking by children 9 years of age or older, city of residence, parental education, and type of cooking fuel used in the home.

Methods

Data were analysed by an approximate *growth curve* method,[14] in which each individual's serial measurements are summarized by a polynomial in time.

The height measurements of each child were expressed as deviations from age-and-sex-specific expected values by subtracting from the observed height the age-and-sex specific predicted height (50th percentile) given by the National Center for Health Statistics (NCHS) growth standards.[15] Each child's mean *level* during the period studied was calculated by averaging these deviations, and the child's *growth rate* was calculated as the slope of a least squares linear regression ($y = \beta_0 + \beta_1 x$) of these deviations on the ages at measurement. The *level* and *growth rate* then became the dependent variables for further regression analyses.

Predictor variables included the proportion of time exposed to gas cooking fuel (0 to 1) and the mean daily tobacco consumptions of the mother and father over the period of observation. Because previous analyses have shown that height and its rate of growth vary among cities, city of residence was included as a set of indicator variables in each analysis, along with a three-level index of socioeconomic status (SES) based upon average parental education (less than 9 years/9 to 12/greater than 12). For the results shown in Tables 1 and 2, each parent's smoking status was entered into multiple regression analyses as a categorical variable with three levels. Results were expressed for smoking groups as the deviation from the effect of the non-smoking group. Sample size was reduced in these analyses because of the requirement for complete data on all covariates. A total of 224 children reported that they smoked cigarettes themselves. Exclusion of these from the following analyses had no effect upon the results.

RESULTS

There was a clear and significant trend ($p<0.0001$) in level of height in children grouped by the number of cigarettes smoked daily by mother (0, 1 to 9, 10+; Figure 1). This negative association between maternal smoking and attained height of child was tested in a multiple regression model adjusting for potential confounding factors (city of residence, SES, father's smoking and cooking fuel) whereupon the association remained statistically significant (Table 1). Adjusting for these factors, we find that children of light smokers have a 0.45 cm deficit and children of heavy smokers a 0.65 cm deficit in attained height. Although non-significant, father's cigarette consumption also showed a negative association with child's height. When the regression model was fitted separately in each of 12 sex by city subgroups (Table 2), children of smoking mothers were nearly always shorter than children of non-smokers, and the findings within each city were generally consistent. However, differences in the fitted model among the 12 subgroups were not statistically significant.

Rate of change in height was not significantly associated with either maternal (bottom of Figure 1, Table 1) or paternal smoking habits (Table 1). Only city of residence was important in predicting growth rate ($p<0.0001$). Subsequent inclusion of initial height (adjusted for age and sex) as a predictor variable, in an attempt to control for parental heights and cumulative prior exposures to the products of cigarette smoking, did not substantially alter these results.

These analyses were repeated using a *per cent of predicted* age-and-sex-adjustment for height with essentially the same results.

DISCUSSION

These results provide additional information regarding the association between passive smoking and height. Although children of smoking mothers are *shorter* in the 6- to 11-year-old age group, inhalation of side-stream smoke is not affecting their *growth rate*. These

TABLE 1 *Regression analyses of 7810 children's attained height and growth rate.**

	Maternal smoking Estimated effect (SEM) for: non-smoker	light smoker	heavy smoker	p
Adjusted height level	0 cm	−0.45 cm (0.23)	−0.65 cm (0.15)	0.0001
Adjusted height growth rate	0 cm/yr	−0.045 cm/yr (0.04)	+0.01 cm/yr (0.02)	0.47
	Paternal smoking Estimated effect (SEM) for: non-smoker	**light smoker**	**heavy smoker**	**p**
Adjusted height level	0 cm	−0.04 cm (0.21)	−0.10 cm (0.15)	0.79
Adjusted height growth rate	0 cm/yr	−0.009 cm/yr (0.03)	+0.005 cm/yr (0.02)	0.92

* Each child's level and growth rate are adjusted for age and sex. City of residence, socioeconomic status, maternal and paternal smoking, and cooking fuel use are all included in the regression models as predictor variables. Sample size is smaller than reported in text because 1463 children have missing values for paternal smoking, socioeconomic status or cooking fuel.

TABLE 2 *Regression analyses of 7810 children's attained height, by sex and city of residence.**

	Estimated effect (SEM) of maternal smoking: non-smoker	light smoker	heavy smoker	N
Watertown boys	0 cm	−0.05 cm (0.55)	+0.07 cm (0.71)	588
Watertown girls	0 cm	−1.51 cm (0.59)	−1.16 cm (0.77)	566
Kingston–Harriman boys	0 cm	−0.33 cm (0.65)	−0.48 cm (0.90)	487
Kingston–Harriman girls	0 cm	−0.29 cm (0.66)	−1.35 cm (0.96)	454
St Louis boys	0 cm	−0.35 cm (0.71)	−0.14 cm (0.45)	780
St Louis girls	0 cm	+0.21 cm (0.80)	−1.01 cm (0.47)	745
Steubenville boys	0 cm	−0.51 cm (0.84)	−0.21 cm (0.48)	711
Steubenville girls	0 cm	−1.32 cm (0.81)	−0.63 cm (0.51)	729
Portage boys	0 cm	−1.41 cm (0.74)	−1.59 cm (0.48)	741
Portage girls	0 cm	+0.67 cm (0.80)	−0.97 cm (0.53)	713
Topeka boys	0 cm	−0.45 cm (0.91)	−0.16 cm (0.50)	672
Topeka girls	0 cm	−0.29 cm (0.83)	−0.28 cm (0.55)	624

* Model was fitted separately to each of 12 subgroups. Other predictor variables in the regression model included socioeconomic status, paternal smoking and cooking fuel use in the home. Sample size is smaller than reported in text (see Table 1).

findings suggest that smoke exposure at a younger age, either during earlier childhood or *in utero*, may be an important determinant of attained height. Since children's height tracks strongly between 6 and 10 years of age,[16] further exposure at these ages apparently has little *additional* effect on height.

Some readers have argued that there was a very small chance of finding an association between passive smoking and height growth rate due to the strong tracking of height measurements in this age range. However, the tracking of height in preadolescent children is only sightly greater than that of FEV_1, after taking measurement error into account. Dockery and colleagues[16] estimated that the tracking index for height along age was 0.95 for girls and 0.97 for boys, and the tracking index for FEV_1 along height (taking into account its larger measurement error) was 0.86 for girls and 0.95 for boys. Despite the strong tracking of FEV_1, Tager *et al*[21] have demonstrated that the growth rate of FEV_1 in children 6 years of age and older is affected by passive smoking.

The association between maternal smoking and height of offspring is also unlikely to be explained by a tendency for shorter women to smoke with greater frequency and to have shorter children. Although height had not been recorded for the mothers of participating children, an analysis of height and smoking behaviour among a random sample of 1630 women between the ages of 25 and 44 and living in the same six cities indicated that (controlling for education, city of residence and age) women who are 1 cm taller are estimated to smoke on the average 0.06 of a cigarette more per day ($p = 0.27$).

These results are not explained by mothers of lower

SES being heavier smokers *and* children of these mothers receiving less adequate nutrition, since analyses controlled for socioeconomic status. Both SES and city had highly significant regression coefficients in the analysis of attained height. These factors could be markers of potentially important risk factors such as dietary differences, illness rates, or other factors which have been studied by other authors.[10]

There was no significant difference ($p = 0.77$) in height between children who remained in the study and those who do not appear in these analyses because they dropped out after the initial visit. Thus there is no evidence of selection bias which could be responsible for our results.

Although two previous studies[9,12] also reported a dose-response association with the same maternal smoking categories, our estimates generally are smaller than those reported by other authors.[9–11] Most of these studies had ample sample sizes (one had over 3000; another over 10000 children) and all made attempts to control for associated sociological and biological factors. Our smaller estimate would be expected if the effect of passive smoke exposure on height occurs during earlier childhood or pregnancy, for the parental smoking variable in our analyses measures the current exposure of the school-age child rather than the early childhood or fetal exposure. Our distribution (Figure 1) of 53% non-smokers, 9% light smokers and 37% heavy smokers represents a somewhat higher percentage of heavy smokers than reported from studies of pregnant women.[9]

Our results could be interpreted to imply that differences in height resulting from maternal smoking during pregnancy persist up to the onset of puberty. This would suggest that there is little catch-up growth, contrary to what is often observed in other phenomena such as recovery from early malnutrition. Smith *et al*[19] and Berkey *et al*[20] demonstrate that infants frequently change length and weight percentile levels as they grow and do not *track* along the growth charts until after age 18 or 24 months. Therefore we would expect that *sustained exposure* to the products of smoking at least throughout the first year of life would be required to produce an effect still observed in 6-year-olds. Unfortunately, we do not have available in our study the data required to test this hypothesis.

Rona *et al*[17] found a significant dose response relationship between number of *current* smokers in the home and height of elementary school children. They controlled for each child's birthweight (as well as parental heights, number of siblings and father's social class) and thus concluded that the association between number of smokers in the home and height cannot be explained by maternal smoking during pregnancy, but instead by later passive smoking. Their maximum mean difference between groups was 1 cm. Studies by Rona and Florey[18] indicate that respiratory illness is not a mediating factor between passive smoking and height, for the association between respiratory symptoms and height was marginal.

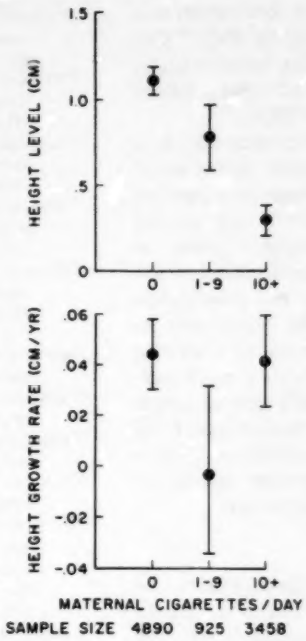

FIGURE 1 *Within maternal smoking categories, means of height level and growth rate. Each child's level and growth rate were previously adjusted for age and sex. Vertical bars are* ± SEM (*standard error of mean*).

The statistically significant association between attained height and exposure to maternal smoking but not exposure to paternal smoking is not surprising. Most studies of passive smoking and respiratory health in children also note larger effects from maternal than from paternal smoking. This is generally explained as some combination of an effect of smoking during pregnancy and the tendency for young children to spend more time with their mothers.

A special concern for authors of studies of pulmonary function in children is that pulmonary function is typically adjusted for body size. All methods proposed to date for adjusting for body size have used some function of height. As a result, subsequent analyses will provide conservative estimates of the effects of passsive smoking on lung function.

Note that a height deficit of 0.6 cm corresponds to a deficit of 18 ml for FEV_1 and 22 ml for FVC.[16] Our research in progress on lung function values indicates that, unlike height, exposure to sidestream smoke affects *both* level and growth rate of FEV_1.

In summary, smoking during pregnancy may be a large component in the observed height deficiency of 6-to 11-year-olds, but sustained exposure to sidestream smoke in the infant and young child may prevent complete catch-up growth that might otherwise be observed. Since we do not have smoking information on the parents before the onset of our study (when children were under 5 years), we are unable to investigate this issue directly. But we were able to demonstrate that passive smoking in the 6- to 11-year-old child does not affect the child's rate of height growth. Future analyses of data collected from these children during adolescence will determine whether these height differences persist beyond puberty or diminish during the adolescent growth spurt.

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